A CONTRIBUTION TO THE CLINICAL STUDY OF SPONTANEOUS DEGENERATIVE NEURITIS OF THE BRACHIAL PLEXUS.[1]

By WILLIAM M. LESZYNSKY, M.D.,

Lecturer on Mental and Nervous Diseases at the New York Post-Graduate Medical School; Attending Physician in the Department for Nervous Diseases at the Demilt Dispensary, etc.

WITHIN recent years the literature relating to peripheral neuritis has indeed been voluminous, particularly that pertaining to the multiple form. Hence, much that is definite and decided has been added to our knowledge of this affection, which, to-day, is universally recognized and firmly established.

This paper is intended to serve as a contribution to the clinical study of spontaneous neuritis affecting the brachial plexus, and is based upon the observation of the following unique case:

Michael Carney, an Irish laborer, thirty-eight years of age, first came under observation on the 15th of October, 1887, complaining of pain in the *left* shoulder, of one week's duration. He describes the character of the pain as constant, "shooting" down the inner aspect of the arm to the elbow-joint, and is apprehensive in regard to the probably serious nature of his malady, as three years ago he suffered from an attack of paralysis affecting the *right* shoulder, which began in a similar manner and resulted in six weeks of enforced idleness. His former trouble, he believes, was due to "taking cold," and the cause of his present illness he ascribes to a thorough drenching experienced in a rainstorm a few days before existing symptoms began. He has been married seventeen years, and has four children living. His wife states that she has had no miscarriages, but four of her children died from convulsions during infancy. Repeated interrogation fails to elicit any antecedent history of traumatism, syphilis, or joint disease. He confesses

[1] Read at the Annual Meeting of the American Neurological Association, June, 1889.

excessive indulgence in alcoholics, followed frequently by intoxication, but has abstained from the use of liquor during the last two weeks. Some years ago he had occasional attacks of muscular rheumatism (lumbago, pleurodynia, etc.). Bowels are constipated and appetite is poor.

Status Præsens.—Well-nourished, able-bodied man, with a moderate degree of intelligence.

Left Upper Extremity.—Motility unimpaired; muscular resistance good; no roughening or involvement of shoulder-joint; tenderness and circumscribed pain on pressure over lower portion of biceps; no objective sensory disturbance. Dynamometer registers 80 (right, 100+). All nerves and muscles react well to faradism, secondary coil, slide at 12 mm.

Right Upper Extremity.—Deltoid is markedly atrophied. Muscular resistance is excellent. He has full use of the shoulder and arm, the loss of the deltoid being replaced by the compensatory development of the other shoulder muscles. The extremity is otherwise normal. The deltoid does not react to faradism. Galvanic current of 15 milliampères produces feeble CaCC.

Lower extremities normal. No evidence of cardiac, pulmonary, or renal lesion.

Treatment.—Abstinence from alcoholics. Blister over biceps. Calomel, gr. v; compound licorice powder, ʒ i: at one dose.

Oct. 20, 1887. Reports to-day that the calomel, etc., produced free purgation, and that he has been in constant pain, which begins in the left shoulder and extends to the biceps. The pain is worse at night, and he is unable to sleep. The *left deltoid is paralyzed*, and there is some difficulty in outward rotation of humerus. No objective sensory disturbance.

Electrical Reaction.

	Faradism.	*Galvanism.*
L. circumflex nerve,	O.	O.
L. deltoid, 12 mm.,	good.	5 Ma.CaCC.

Ordered rest, blisters, and antipyrin twenty grains every hour for three hours, unless relief is obtained sooner.

Oct. 25th. Pain is paroxysmal and almost unendurable, darting down to the dorsum of thumb and index-finger. He is unable to sleep at night, and "walks the streets" endeavoring to obtain relief through exercise. Repeated doses of antipyrin proved ineffectual.

Slight temporary alleviation was obtained by wrapping the arm in hot-water cloths. He states that when the right arm was affected, the pain was just as severe, but did not extend below the elbow, and was always worse at night. He believes the arm and hand are getting weaker, and complains of numbness below the shoulder, extending over the radial distribution. Dynamometer registers 65 (R., 100+). There is some tenderness over the lower portion of the radial nerve, but no appreciable thickening. In the region of the cutaneous distribution of the circumflex nerve there is an area, 2½ cm. wide and 7½ cm. in length, where tactile, pain, and temperature senses are abolished. The numbness is greatest at this point. Temperature in mouth, 99⅘° F.

Electrical Reaction.

Farad.	*Galv.*
Deltoid, 12 mm., feeble.	5 Ma.CaCC, slow.
Musculo-spiral nerve and muscles, 15 mm., good.	

He refuses to remain abed, saying that he is more comfortable while walking about. Ordered morphia sulphate, one-sixth grain, and repeat if necessary; blisters; salicylate of soda, twenty grains every three hours.

Oct. 29th. Pain was relieved by one third grain of morphia. Slept well last night without medicine. The hand is weaker. Grasp with dynamometer, 38. The biceps and triceps exhibit well-marked fibrillary tremor. In the fibres, at the lower portion of these muscles, there occur distinct, irregular, and wave-like contractions, forming "myoid tumors" parallel with the course of the muscular fibres. These circumscribed contractions are produced by either voluntary or passive motion or mechanical irritation. There is partial wrist-drop, the extensors of the forearm and fingers and the supinators being paretic. There is also

difficulty in outward rotation of the arm, indicating the involvement of the infraspinatus and teres minor.

The pectoralis major and the biceps group are weak, but the triceps is not affected. Area of anæsthesia unchanged. Temperature in mouth, 100° F.

Nov. 1st. Was comparatively comfortable until yesterday. Then pain began at elbow and extended over radial side of forearm to the thumb and index-finger, but is now more intense over the dorsum of thumb. Recumbency increases the pain, which is invariably worse at night. Complete *paralysis* of *infraspinatus* and *teres minor* (no outward rotation). Inward rotation of humerus good. The *pectoralis major*, *supinators* and *extensors* of *forearm* are also paralyzed. Triceps apparently not involved, but "jerk" is feeble. Dynamometer, 48; temperature, 99° F. Discontinue salicylate of soda. To have hydrarg. bichlorid., gr. $\frac{1}{24}$ t. i. d.

Electrical Reaction.

	Faral.	*Galv.*
Deltoid 12 mm. feeble.		
Musculo-spiral n. 15 mm.		
Extensors, O.		5¼ Ma. CaCC. feeble.

No reaction can be obtained at "Erb's Supraclavicular point" on either side.

Nov. 3d. On the night of November 1st pain was excruciating and constant, darting up and down the arm in the course of the musculo-spiral and radial nerves to the thumb and index finger. Took ⅔ grain of morphia without relief. Pain began at 6 P. M., and ceased at 6 A. M. He slept well last night without morphia, and the pain has subsided. The grasp is weaker. Dynamometer 25. The *triceps* is *paralyzed* and "jerk" is abolished. There is a point of extreme tenderness on deep pressure just above the flexure of the elbow joint. Analgesia in the course of the radial distribution to the hand. Partial tactile anæsthesia, more marked over the dorsum of the thumb. Area of anæsthesia below deltoid unchanged.

Electrical Reaction.

Faradism.

	RIGHT.	LEFT.
Musculo-spiral N.,	15 mm. fair	O
Triceps,	"	O
Deltoid,	O	O
Extensors,	15 mm. fair	O
Median and Ulnar N. and Muscles,	Normal.	Normal.

Galvanism.

	RIGHT.	LEFT.
Musculo-spiral N.,	8 Ma. CaCC *slow.*	O
Triceps,	6½ Ma. CaCC> AnCC.	3½ Ma. CaCC> AnCC *D. T.*
Deltoid,	15 " CaCC feeble.	3 " CaCC> AnCC *slow.*
Extensors	7 " CaCC> AnCC.	3½ " " " "
Median and Ulnar N. and Muscles,	Normal.	Normal.

Nov. 5th. No pain since last note. Only slight aching in elbow. Grasp is stronger. Dynamometer 38. General condition of extremity unchanged. During the last few days he felt occasional slight pain, accompanied by tremor over the right serratus magnus.

Measurement.

Circumference.

	RIGHT.	LEFT.
Arm, 6 in. below acromion (extremity pendent)	10¾ in.	10½ in.
Forearm, 4 in. below olecranon (" semi-flexed)	10½ "	10½ "

Nov. 8th. Slept well on the 5th without morphia. Pain returned the following night. *Causalgia* affecting the dorsum of hand during the past week. On the 7th, had pain all day, mostly at the flexure of the elbow-joint, over the lower end of the biceps, with continued numbness in the course of the radial nerve. Last night suffered from severe pain, stabbing, shooting and darting in character. He obtained some relief from the application of hot-water cloths. There is a slight œdema over the dorsal aspect of hand.

Internal rotation of *humerus abolished* (indicating involvement of the subscapular nerves). Dynamometer 32. Sensory disturbance, etc., same. No pupillary symptoms.

Nov. 10. Pain has extended over the forearm. Anal-

gesia and partial tactile anæsthesia over the radial and external cutaneous branches of the musculo-spiral nerve. *The biceps* is *paretic*. With great effort he succeeds in slightly flexing the forearm. Pain is aggravated by passive extension of the forearm, owing to deep-seated inflammation, probably of the musculo-spiral nerve. Slight pressure at the flexure of the elbow produces severe pain, which radiates over the extensors. (I was unable to satisfactorily determine the existence of thickened or swollen nerve).

Electrical Reaction.

	Farad.	*Galv.*
External Cutaneous N.,	12 mm.	6 Ma. CaCC.
Biceps Group,	" fair.	" CaCC=AnCC.

Nov. 19th. Did not appear since last note until to-day. Remained abed until yesterday as he felt weak. No pain. Only numbness, as usual. Slept well. Upon leaving his bed, the pain returned in the shoulder (stormy weather). Pain is paroxysmal, and was so severe last night that he was unable to sleep. Constant causalgia over the hand and the wrist-joint. When the paroxysm of pain comes on, it is accompanied by "weakness" in the præcordial region, with a feeling of faintness, and then "doesn't care whether he dies or not." Is relieved by a full inspiration and "fixing" chest. No pupillary symptoms. Scapular muscles and deltoid undergoing atrophy. Biceps group weaker. Flexion of hand and fingers good. Oedema over the dorsum of the hand increasing. Dynamometer 33. Analgesia over the entire course of the external cutaneous and radial nerves, with partial tactile anæsthesia and loss of temperature sense.

Electrical Reaction.

	Farad.	*Galv.*
Musculo-spiral nerve,	O	O
Triceps,	O	5 Ma. CaCC> An CC *slow.*
Deltoid,	O	5 " AnCC> CaCC *feeble.*
Extensors,	O	5 " CaCC> AnCC *slow.*
Biceps Group,	12 mm. feeble.	
Supra and infraspinati,	O	

Median and ulnar nerves and muscles react well to faradism 17 mm.

Continue hydrarg. bichlorid $\frac{1}{24}$ t. i. d.

Nov. 22d. Location and severity of pain unchanged. *Biceps group paralyzed.* Dynamometer 30.

Measurement. Circumference of arm 10¼ in. Forearm 9½ in. Loss, ¼ and 1 in.

Electrical Resistance. *See p.* 33 } Right upper extremity, 1700 ohms. Left " " 2280 "

Being 580 ohms greater in the affected arm.

Nov. 26. He was obliged to take two doses every night to relieve the pain, which also continues during the day. This morning he awoke with pain, as usual, which was worse in the course of the radial nerve, and affected the thumb and index finger. Causalgia continues. Trophic changes in the course of the radial distribution over the thumb and index finger. The skin is pale, glossy, œdematous and anæsthetic. Dynamometer 20. Atrophy of scapular muscles and extensors of forearm increasing. Anæsthetic area unchanged. Extremely sensitive point over the pectoralis major muscle on a line three inches above the nipple. *The muscles supplied by the median and ulnar nerves are not affected.*

Electrical Reaction.

	Farad.	*Galv.*
Extern. Cutan. N.	13½ mm. *slow.*	6½ Ma. CaCC=An CC.
Biceps Group,	" "	" "

Median and Ulnar Nerves (and Muscles), Normal.

Dec. 1st. During the last three nights has been free from pain, which is now confined to the dorsum of the thumb and index-finger. Dynamometer, 22. Extreme tenderness over biceps and pectoralis major.

Electrical Reaction.

	Farad.	*Galv.*
Mus. spiral nerve, - -	O.	O.
Triceps (scap., head), -	O.	6 Ma.CaCC, slow.
" (short, head), -	O.	6 Ma.AnCC>CaCC, slow.

Extensors, - - - - - O.	8½ Ma.AnCC>CaCC, slow.
Scapular muscles, - - O.	6 Ma.CaCC>AnCC, "
Deltoid, - - - - - - O.	6 Ma.AnCC>CaCC, "
Biceps group, 13½ mm. (fair).	9 Ma.AnCC>CaCC, "
Median and ulnar nerves and muscles, 14 mm., - - -	5½ Ma.CaCC, *normal.*

Dec. 10th. Very little pain until this 3 A. M. (raining). Causalgia constant. Some ability in inward rotation. No outward rotation. Muscular atrophy increasing. Sensory disturbance same. Some hyperæsthesia in palm. No change in motility. Dynamometer, 20. Tenderness over the biceps and pectoralis major unchanged. Administration of hydrag. bichlorid., gr. $\frac{1}{24}$, t. i. d., continued.

Electrical Reaction.

	Farad.	*Galv.*
Musc. spiral nerve, -	O.	O.
Triceps, - - - - -	O.	10 Ma.CaCC>AnCC, slow.
Extensors, - - - -	O.	6½ " " "
Deltoid, - - - - -	O.	10 " " "

Dec. 17th. Tenderness over the pectoralis major and biceps has disappeared. Otherwise unchanged. Dynamometer, 23.

Electrical Resistance.—Right = 5590 ohms. Left = 6480 (dif. = 890).

Electrical Reaction.

	Farad.	*Galv.*
Musc. spiral nerve, - -	O.	O.
Triceps, - - - - -	O.	6 Ma.CaCC>AnCC, slow.
Extensors, - - - -	O.	" CaCC=AnCC, "
Deltoid, - - - - -	O.	" CaCC>AnCC, "
Biceps group, 15½ mm. (slow), - - - - -		" AnCC>CaCC, "
Median and ulnar nerves, 13½ mm., - - - -		" CaCC, normal.

Measurement.—Circumference, arm, 9¾ in.; forearm, 9½ in. (loss, ½ in.).

Dec. 29th. Some improvement in the strength of the biceps. Fibrillary contractions and tenderness over biceps. No other change.

Electrical Resistance.—Right = 5400 ohms. Left = 6300 (dif. + 900).

Electrical Reaction.

	Farad.	*Galv.*
Musc. spiral nerve, - -	O.	O.
Triceps, - - - - - -	O.	8 Ma. CaCC > AnCC, slow.
Extensors, - - - - -	O.	4 " = " "
Deltoid, - - - - - -	O.	8 " " " "
Biceps group, 14 mm. (slow).		5 " " " "
Scapular muscles, - -	O.	8 " > " "

At his request he was sent to Bellevue Hospital.

Feb. 7, 1888. He left the hospital to-day. Since last note had very little pain, which was limited to the hand. He has regained some power in the arm. The only perceptible improvement is in the biceps group. Area of analgesia has diminished in the district of the circumflex, but is still well defined. Tactile sensibility improved. No loss of muscular sense. Trophic changes in the hand are markedly diminished. Atrophy of the scapular muscles increasing. General health good. Sleeps well. Shoulder-joint relaxed and articular surfaces roughened.

Electrical Reaction.

	Farad.	*Galv.*
Musc spiral nerve, - - -	O.	O.
Triceps, - - - - - - -	O.	8 Ma. AnCC > CaCC, slow.
Extensors, - - - - - - -	O.	5 " CaCC > AnCC, "
Deltoid, - - - - - - -	O.	8 " AnCC = CaCC, "
Biceps group, - - -	12½ mm.	4½ Ma. CaCC > AnCC, "

Feb. 9, 1888:

Electrical Resistance.—Right = 5200 ohms. Left = 7000 (dif. + 1800).

March 10th. Since last note suffered occasional nocturnal paroxysmal pains below elbow-joint in radial distribution. He has been a flagman on the Long Island Railroad during the last three weeks. Exercising the affected

arm produces swelling and pain over the wrist-joint and soreness over the pectoralis major. General health good. Has abstained (?) from the use of liquor. Atrophy of shoulder muscles has increased. No improvement in deltoid, triceps, or extensors of forearm. Biceps group improving. Dynamometer (stiffer spring), L. = 27; R. = 100 +. Area of anæsthesia decidedly diminished above elbow-joint. No loss of muscular sense. No trophic disturbance save in dorsal aspect of thumb. Roughening of articular surfaces of shoulder-joint.

Electrical Resistance.—R.= 4700 ohms. L. = 6300 (dif. + 1600).

Electrical Reaction.

	Farad.	*Galv.*
Musc. spiral nerve, - - -	O.	20 Ma. produces feeble CaCC in triceps.
Triceps (outer head), 15½ mm. (feeble), - - - - - - -		
Extensors, - - - - - -	O.	8 Ma. CaCC>AnCC.

Measurement.—Circumference, arm, 9¼ in.; forearm, 9 in.

April 14th. Complains of sudden tremors, localized in spots over the arm and extending to the hand. He says the same thing occurred in the other hand before recovery. No outward rotation of humerus. Very feeble inward rotation. The only improvement in motility is in the biceps group. With great effort he succeeds in producing partial flexion of forearm. No vaso-motor disturbance. No trophic changes in hand. Tactile anæsthesia in spots in the course of the radial nerve only. Area of analgesia limited to two or three spots over the radial distribution at the wrist. Shoulder-joint not so rough as at last examination.[2] Atrophy of scapular muscles unchanged. Dynamometer, 30.

[2] He was recently shown by me at a meeting of the New York Neurological Society (April 3, 1888), at a time when the shoulder-joint was "roughened," owing to paralysis of the shoulder muscles and consequent disuse of the limb. This led one of the members present to suppose that this condition of the joint may have existed prior to the development of the neuritis.

Electrical Resistance.—Right = 5500 ohms. Left = 4330 (dif. — 1170).

Electrical Reaction.

	Farad.	*Galv.*
Musc. spiral nerve,	O.	O.
Supra- and infraspinati,	O.	7 Ma. CaCC, feeble.
Deltoid,	O.	7¼ Ma. CaCC=AnCC, slow.
Triceps,	O.	6 " " " "
Biceps group,	14½ mm.	5½ " " " "
Extensors,	O.	9 " " " "
Median and ulnar nerves and muscles,	14½ mm.	Normal.

April 28th. *Electrical Resistance.*—Right = 5400 ohms. Left = 4500 (dif. — 900).

June 7th. No pain during the last three months. Dynamometer, 33. Roughened shoulder-joint and atrophy unchanged. Slight improvement in inward rotation. No outward rotation. Biceps group have recovered with good resistance to passive motion. Supination of hand is accomplished by the biceps only. No other improvement in motility. The dorsum of the hand is œdematous. Anæsthesia is limited to one small spot over the dorsum of the thumb. The entire surface of the extremity is perspiring, save over the radial distribution below the wrist, where the skin is dry.

Measurement.—Circumference, arm, 9¼ in.; forearm, 9¼ in.

Electrical Reaction.

Faradism.

Biceps,	15 mm.

Galvanism.

Deltoid,	7 Ma. CaCC>AnCC,	slow.
Triceps (scap. head),	12 " AnCC=CaCC,	"
Triceps (short head)	8 " CaCC>AnCC,	"
Biceps group,	5 " " " "	"
Extensors,	8 " AnCC>CaCC,	"

Sept. 11th. In rainy weather has occasional pain and numbness in the hand and over the deltoid. Atrophy of scapular muscles, deltoid, triceps, and extensors of forearm. Complete paralysis of the deltoid and extensors. Partial paralysis of the triceps. Inward rotation good. No outward rotation. Biceps group normal. Dynamometer: L., 43; R., 100+. No vaso-motor changes. No sensory disturbance. Area of anæsthesia has disappeared. Roughening at shoulder-joint unchanged. Elbow-joint normal. Complete relaxation of wrist-joint. No roughening.

Measurement. Circumference, arm 9½ in.; forearm, 8¾ in.

Electrical Reaction.

	Farad.	*Galv.*
Musc. cutan. nerve and biceps group	10 mm.	4 Ma.CaCC.
Supraspinatus, -	O.	5 Ma. CaCC. (slow).
Infraspinatus, -	O.	10 " O.
Triceps (short head,	10 mm.	7 " AnCC>CaCC. (slow).
" (scap.head),	O.	10 " " " "
Deltoid, - - -	O.	7 " AnCC=CaCC. "
Extensors, - - -	O.	7 " AnCC>CaCC. "

Median and ulnar nerves and muscles normal.

Dec. 27. Since last note he has been at work driving a horse. The atrophied muscles are improving. Inward rotation good. Partial outward rotation and improvement in the action of the pectoralis major. There is a slight return of power in the deltoid. Has good use of the triceps. Biceps group have remained well. There is a tender point over the radial nerve, producing radiation of pain to the tip of the thumb and index finger.

Measurement. Circumference, arm 9¾ in.; forearm, 9⅛ in.

Electrical Reaction.

	Farad.	*Galv.*
Musc. spiral N, - -	O.	O. - - - - - -
Supraspinatus, -	10½ mm.	- - - - - - - -
Infraspinatus, -	O.	16 Ma.CaCC, (feeble).
Deltoid, - - -	10½ mm.	10 " AnCC>CaCC (slow).
Triceps, - - -	"	10 " CaCC=AnCC.
Biceps group, -	"	- - - - - - - - -
Extensors, - -	O.	20 Ma. No polar reaction; only feeble reaction to labile cathode.

Median and ulnar nerves and muscles normal.

Jan. 31, 1889. No pain since last note. After using the left hand and arm, numbness and fibrillary contractions occur over the dorsum of the hand. The motility is increasing in the shoulder and upper arm muscles. No improvement in the extensors and supinators of forearm.

June 15. He has been steadily employed since last note, and the condition of the left upper extremity is much better. The atrophied muscles are improving. Inward rotation of the humerus is good. Outward rotation is incomplete. The deltoid muscle is weak, but its motility is restored. Triceps and biceps group normal. There is some return of voluntary power in the extensors and supinators of forearm and in the extensor communis digitorum. There is slight roughening of the shoulder-joint.

Measurement. Circumference, arm 10¼ in.; forearm, 9½ in.

Electrical Resistance. R. = 8700 ohms; L. = 6300 (dif. —2400).

Electrical Reaction.

	Farad.	*Galv.*
Infraspinatus, 1 mm. (feeble).		22½ Ma. CaCC>AnCC (feeble)
Deltoid, - 13 mm.		8½ " " "
Mus. spiral N, 1 mm. (feeble).		12½ " CaCC (feeble).
Triceps, - 13 mm.		- - - - - - - - - - - -
Biceps group, "		- - - - - - - - - - - -
Extensors, - O.		19 Ma. AnCC>CaCC (feeble).

Median and ulnar nerves normal.

During the entire period of observation he was extremely irregular in his attendance. Many times it was necessary to send for him to appear for examination. He virtually received no systematic treatment. The only instructions known to have been indifferently followed at home, after acute symptoms had subsided, were passive motion to all joints, daily bathing and manipulation of the muscles, and keeping the limb suspended and protected from exposure.

SUMMARY.

A man, thirty-eight years of age, shortly after exposure to cold (without antecedent history of joint disease or injury), suffered from severe paroxysmal pain in the left shoulder, rapidly followed by paralysis of the deltoid. After short but varying intervals of freedom from acute pain, another paroxysm would occur, accompanied by additional paralyses. These attacks, extending over a period of four weeks, involved all of the muscles innervated by the circumflex, suprascapular, subscapular, musculo-cutaneous and musculo-spiral nerves. There was anæsthesia in the domain of the circumflex, external cutaneous and radial nerves. Well-marked atrophy, with the reaction of degeneration, existed in all of the paralyzed muscles.

Trophic changes were present in the skin over the thumb and index finger.

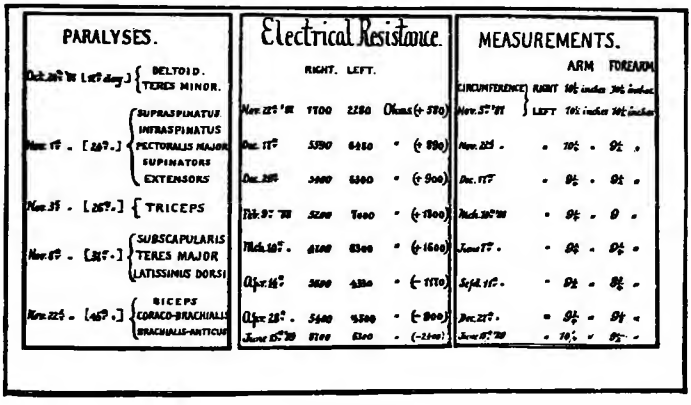
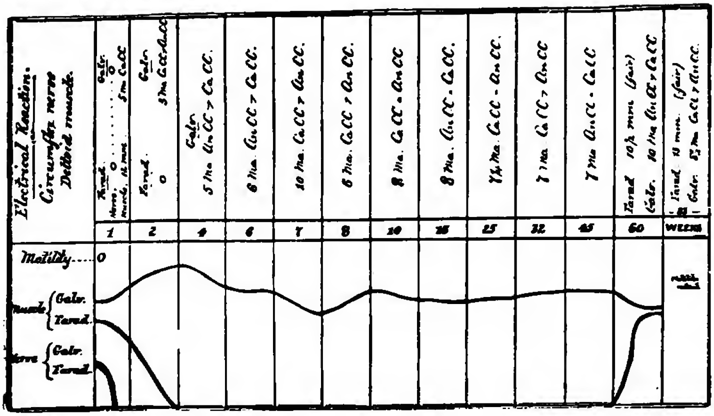
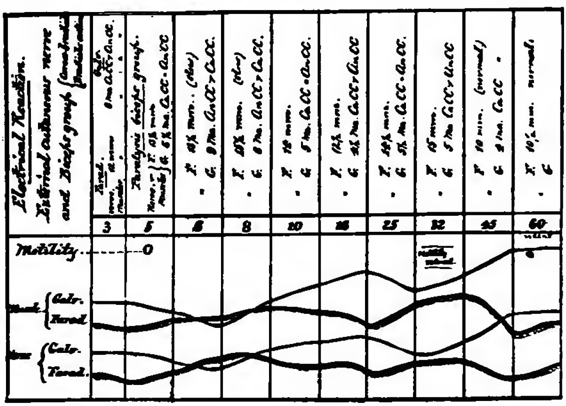
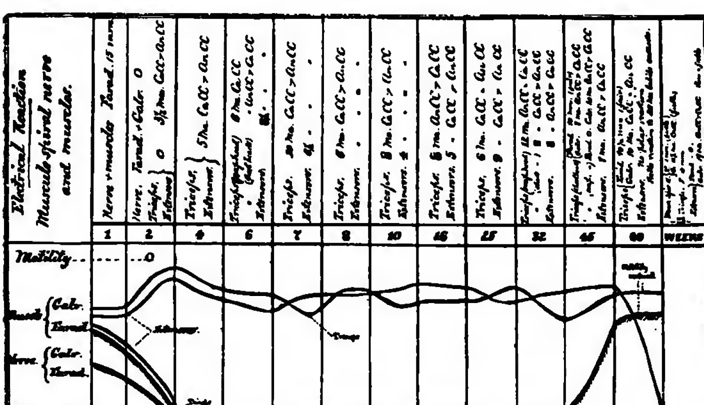


TABLE A.

The median and ulnar nerves were not implicated. Almost complete recovery at the end of two years. It is well known that motor function is always more easily abolished than sensory function, and that in cases of recovery from damage sensation invariably returns before motion. In this instance, the area of anæsthesia began to diminish after four months. At the end of one year all disturbance of sensibility had disappeared.


Electrical Reaction.
Circumflex nerve
Deltoid muscle.
1
2
4
6
7
8
10
16
25
32
45
60
WEEKS
Motility
Muscle
Galv.
Farad.
Nerve
Galv.
Farad.


Electrical Reaction.
External cutaneous nerve
and Biceps group
3
5
6
8
10
16
25
32
45
60
Motility
Muscle
Galv.
Farad.
Nerve
Galv.
Farad.


Electrical Reaction
Musculo-spiral nerve
and muscles.
1
2
4
6
7
8
10
16
25
32
45
60
WEEKS
Motility
Muscle
Galv.
Farad.
Nerve
Galv.
Farad.
Extensors
Triceps

The return of motility took place in the following order, the muscles attacked last being the first to recover (see table).

1st. Biceps group.
2d. Subscapularis and teres major.
3d. Triceps.
4th. Supraspinatus, infraspinatus, teres minor, deltoid.
5th. Extensors and supinators of forearm.

THE ELECTRICAL RESISTANCE.

In obtaining the electrical resistance in the extremity, the following method was adopted: A flat flannel-covered sheet-lead electrode, 2 x 4 inches, being thoroughly moistened with hot salt water, was securely attached over the nucha. A similar electrode, 1 x 2 inches, was firmly fastened in the palmar surface of the hand, the hands and the electrode having been previously soaked in the hot salt solution.

A sufficient number of Leclanché elements were then slowly introduced in the circuit (descending current) until 4 milliampères were registered on the meter. The same process was then applied to the other arm.

Subsequently, the amount of resistance was determined by means of a wire rheostat. A sufficient number of ohms (in place of the patient's body) were introduced in the circuit produced by the same electromotive force used in the examination of the patient until the meter registered 4 milliampères.

The stationary electrode at the neck was kept thoroughly wet. In order to produce uniform pressure in both hands, and to avoid unequal condensation of tissue, the electrode was retained in position by a band.

As there are so many elements of error to be eliminated before we can succeed in obtaining an accurate measurement of the electrical resistance in the human body, I would only claim the measurements in this case to be approximately correct.

In all eight measurements were made, extending over a period of nineteen months (see table A.) Five comparative examinations, during the existence of acute symptoms, invariably revealed a condition of greater resistance in the affected limb. At three subsequent examinations, after the subsidence of all acute symptoms, the resistance was found markedly diminished.

These uniform results are certainly conclusive in establishing the fact that the electrical resistance was increased during the active stage of the disease, and diminished during convalesence.

It will be noted that on November 22d, 1887, the resistance was R=1700 ohms, left 2280; while on June 15th, 1889, it was R=8700 ohms; Left =6300.

That such an apparent discrepancy should exist,can only be explained (in the last observation) on the ground of the probably insufficient moisture of the skin, or the difference in the pressure of the electrodes as compared with previous examinations. Despite this great difference in the resistance from time to to time, the uniformity of the comparative variations, at each measurement, is sufficiently suggestive to warrant further investigation along this line. These observations are merely tentative, and under existing circumstances cannot be considered of diagnostic significance.

Before analyzing the salient features of this case, it would be well in this connection, to present a brief resumé of our knowledge of the anatomy and physiology of the brachial plexus. It is beyond the scope of this paper to enter at length upon a discussion of this subject.

Quite a diversity of opinion seems to exist as to the gross anatomy of the brachial plexus. Gray[3] states, that "the fifth and sixth cervicals unite near their exit from the spine into a common trunk; the seventh cervical joins this trunk near the outer border of the middle scalenus; the three nerves thus form one large single cord. The eighth cervical and first dorsal nerves unite behind the anterior scalenus into a common trunk. Thus two large trunks are formed,

[3] Anatomy Descriptive and Surgical, 1871, p. 638.

the upper one by the union of the fifth, sixth and seventh cervicals; and the lower one by the eighth cervical and first dorsal. Opposite the clavicle, and sometimes in the axilla, each of the cords gives off a fasciculus, by the union of which a third trunk is formed."

Heath, Leidy, Quain, Ellis and Flower give the same arrangement, while the arrangement furnished by Sappey, Cruveilhier, Hirsehfeld, Henle, Hyrtl, Longet and Lucas differs mainly from the preceding in that the seventh does not join the common cord of the fifth and sixth, but runs as a separate trunk, dividing below as do the other trunks. (Walsh). Other writers to the number of thirty hold different views in regard to the formation of the plexus.

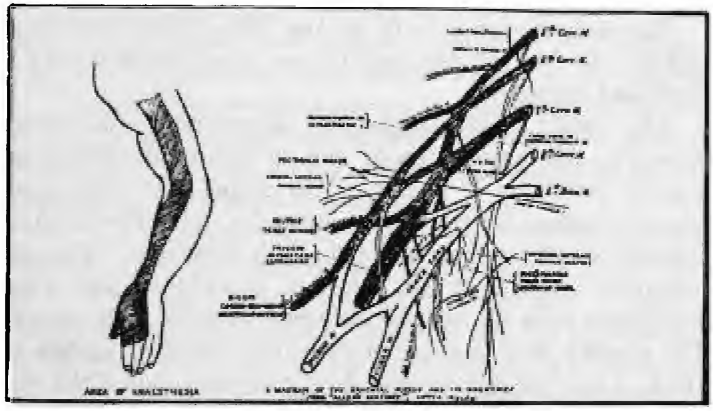


CHART B.

According to the researches of Walsh[4] the arrangement of the plexus is not so variable, as most of the variations can be artificially produced. As a result of his examination of 350 plexuses, he concludes that "nearly every plexus will be found to resolve itself into one and the same arrangement, (see chart B) and it will also be discovered that most of the numberless variations to be found in anatomical works are nothing but normal arrangements distorted by wrong dissections." In regard to the distribution of the filaments composing the five primary trunks to

[4] Amer. Jour. Med. Sciences, Oct. 1887.

the terminal branches of the plexus, the same writer makes the following statement, based upon the study of 74 plexures dissected after maceration in dilute nitric acid.[5]

The *musculo-cutaneous* was supplied by the fifth and sixth in 50 cases, in 23 by these and the seventh, and in one (an anomalous plexus) by a few filaments from the fourth and by the fifth and sixth. The *median* in 66 by all five, and in 8 by the four lower. The *ulnar* in 71 by the seventh and eighth and first dorsal, in two by the two latter, and in one (same anomaly as above) almost entirely by the seventh a few fibres being furnished by the eighth. The *Circumflex* in 63 by the fifth and sixth, in ten by the fifth, sixth and seventh, and in one (same anomaly) by the seventh and eighth.

The *musculo-spiral* in 67 by the fifth, sixth, seventh and eighth. In 6 by all five, and in one (same anomaly) by the sixth and seventh.

The result of Herringham's[6] dissection of the brachial plexus in the human fœtus and in the adult differs only slightly from that of Walsh. He claims that "the *median* nerve is formed by two heads, into the outer the sixth and seventh always enter, while the fifth does not. The inner is formed always by branches of the eighth and ninth,[7] sometimes with the addition of some bundles of the seventh. The median then is made of the sixth, seventh, eighth and ninth. The sixth bundle runs down the outer side of the nerve from the top to the bottom. The supply of the fifth by its anterior branch ends therefore with the musculo-cutaneous nerve. The eighth and ninth usually supply the flexor sublimis and always the deep flexors. After the forearm muscles have been supplied, the remainder of the median which comes from under the flexor sublimis always contains fibres from the sixth, seventh and eighth roots and sometimes a bundle from the ninth. The most common origin

[5] Minute dissection and maceration of the brachial plexus in dissociating liquids was done by W. Krause many years ago. Beitrage Zur Neurologie der oberen Extremitat, 1865.

[6] Proc. Roy. Soc. Lond. 1886--xli- 423.

[7] He calls the first dorsal nerve the ninth spinal root.

of the *ulnar* nerve is from the eighth and ninth together. The *circumflex* is derived from the fifth and sixth alone. The *musculo-spiral* is sometimes formed by all four upper roots, usually by the sixth, seventh and eighth alone.

From a physiological standpoint, the experiments of Ferrier and Yeo[8] command attention. They found that in the brachial plexus in monkeys, no *ulnar* movement occurred when any of the anterior roots above the first dorsal were stimulated. No *musculo-cutaneous* movement followed stimulation of any roots below the sixth cervical; but both *median* and *musculo-spiral* movement followed stimulation of the sixth, seventh and eighth cervical and first dorsal nerves.[9]

Gowers[10] expresses the opinion that we are not justified in transferring these facts to man, except in so far as they receive confirmation from human anatomy and pathology. Nevertheless, much has been added to our knowledge of the function of the brachial plexus by the study of tumors, such as neuromata, and surgical as well as other injuries affecting its branches, which to a great extent favors the confirmation of Ferrier's experiments.

The clinical observations of Erb[11] have served to throw additional light upon the localisation of lesions in the brachial plexus. In the year 1874, he described a form of paralysis arising spontaneously as well as from traumatism, affecting simultaneously the deltoid, biceps, brachialis anticus and the supinator longus. Occasionally the supinator brevis, and at times all the muscles supplied by the median nerve are coincidently involved. The first-named group of muscles, however, are always chiefly if not exclusively affected. He believes the seat of the lesion to be at a point where the fibres forming the circumflex, musculo-cutaneous and a part of the musculo-spiral nerves lie in close proximity to each other. He further states that it is

[8] Proc. Roy. Soc. Lond. 1881, xxxii.

[9] Ferrier has since stated (Proc. Roy. Soc. Lond. 1883—xxxv) that the relations he gave were all one nerve too high. I have therefore designated the nerves in accord with his note of correction.

[10] Dis. Nerv. Syst., Am. Ed. 1888, p. 86.

[11] Ziemssen's Cyclop. 11, p. 561.

possible, by very careful Faradic excitation of the several branches of the plexus, to succeed in discovering in many individuals, a spot (which corresponds to about the point of emergence of the sixth cervical nerve between the scaleni) from which the deltoid, the biceps, brachialis anticus and the supinator longus may be thrown into common and very energetic contraction.[12]

He is consequently of the opinion that this form of paralysis has its seat in the root of the brachial plexus at this point. Hoedemaker[13] however, maintains that the lesion is situated in the cord formed by the fifth and sixth cervical nerves, and calls attention to the fact that these two nerves, owing to their superficial position in their exit between the scaleni muscles, are especially exposed to injury.

Additional cases of Erb's paralysis have been recorded by Remak,[14] Bernhardt,[15] Weill,[16] Nonne,[17] Lannois,[18] Girandeau,[19] and others.

From our knowledge, based upon the foregoing anatomical, physiological and clinical data, we are enabled to determine, with some degree of accuracy, the seat of the pathological process in our case.

The distribution of the paralysis corresponds with a lesion limited to the anterior branches of the fifth, sixth and seventh cervical nerves. The escape of the ulnar nerve and the absence of all pupillary phenomena, are indicative of the preservation of the two lower roots of the plexus.

The elaborate experimental and clinical studies of Klumpke[20] show that oculo-pupillary troubles occur in total paralysis of the brachial plexus, but only in those cases

[12] I have repeatedly demonstrated this "supraclavicular point," which is one of the most interesting in electro-diagnosis.

[13] Deutsch Archiv. Klin. Med. v 40, p. 62, 1887.

[14] Berlin Klin. Woch. No. 9—1877.

[15] Zeitsch. fur. Klin. Med., Bd. iv. 3 Heft.—1882.

[16] La Province Medicale, No. 48—1888.

[17] Deutsch Arch. Klin. Med., V. 40, p. 62—1887.

[18] Revue de Medicine, p. 988—1881.

[19] Ibid. p. 186—1884.

[20] Revue de Medicine, 1885—v— p. 739.

where the lower roots are involved, and arise from a lesion affecting a communicating branch of the first dorsal nerve.

The independent investigation of both Herringham and Walsh show that the *median nerve* invariably received fibres from the sixth and seventh cervical nerves. Its escape, therefore, is certainly extraordinary, and can only be accounted for on the ground of its anomalous origin.

It would be difficult to reconcile these facts upon any other hypothesis.

Cases of this character must either be of very infrequent occurrence, or they are not reported, as no analogous instance can be found in medical literature.

The nearest approach to analogy is the following history, reported by J. Straus.[21] A vigorous, muscular man, 33 years of age, upon awaking one morning, without any antecedent history of injury, rheumatism or exposure, found the right hand numb and the arm seemed heavier than usual. Within two days the numbness had extended to the entire arm, with gradual increase of weakness, until all of the muscles of the extremity were paralyzed, save those innervated by the median nerve.

A study of the sensibility confirmed these facts. Anæsthesia existed in the cutaneous distribution of the circumflex, internal cutaneous, musculo-spiral, ulnar and musculo-cutaneous nerves, while sensation was preserved in the entire domain of the median. The paralyzed muscles reacted to faradic and galvanic irritation.

The patient made a complete recovery at the end of seven weeks. The nerves regained their function in the following order:

1st. Musculo-cutaneous (within two days.) 2. Musculo-spiral. 3d. Internal cutaneous. 4th. Ulnar. 5th. Circumflex.

The return of sensation and motility in the course of the musculo-spiral and the ulnar was parallel and simultaneous. It was only in the circumflex that the return of motion pre-

[21] "Note sur un cas de paralysie spontaneé du plexus brachial (avec integrité du nerf median) et sur quelques localisations rarer de paralysie du plexus brachial." Gaz. Hebdom., 1880, No. 16.

ceded that of sensibility. He believed the case to be one of congestive or inflammatory origin, affecting the branches of the brachial plexus. He offered no explanation for the intact condition of the median nerve, and claimed that no analogous case had been reported.

Buzzard[22] refers to a case (without history) of neuritis affecting certain branches of the brachial plexus and occasioning local paralysis, exquisite pains, hyperalgesia, muscular atrophy, abolished or diminished electrical excitability, and trophic changes in the skin.

Althaus[23] instances the case of a girl sixteen, in whom, after ten days of severe pain and numbness in the right hand and arm, the entire extremity became completely paralyzed. This was accompanied by anæsthesia, trophic changes in the skin, and loss of faradic irritability. There was no atrophy.

Diagnosis.—Rheumatic neuritis of the brachial plexus. Recovery from periphery to centre at the end of seven months.

THE ETIOLOGY.

The prominent factors leading to the development of the neuritis in my case, were alcoholism as the predisposing element and exposure to cold the exciting cause. This may or may not have been accompanied by over-exertion. Leyden[24] maintains that the spontaneous or primary form of multiple neuritis is most commonly caused by exposure or over-exertion, and frequently by both combined. The same may be said of the form of neuritis now under consideration. It is evident that these two factors play an important part in the development of the so-called idiopathic neuritis, as they do in diseases of the spinal cord. According to Caspari's observations, cold is one of the most frequent causes of neuritis in Russia, where remarkable temperature variations exist during many months. Since Magnus Huss in 1852 directed attention to a form of paralysis occurring in alcoholic subjects, many important

[22] Lond. Lancet, 1885, vii., p. 983.

[23] Med. Chir. Trans., Lond., 1871, v., 54.

[24] Die Entzündung der peripheren Nerven, Berlin, 1888.

contributions to our knowledge of this matter have been made, so that it now is a well-known and established fact that the toxic effect of alcohol upon the peripheral nerves renders them more vulnerable to affections of an imflammatory or degenerative nature, and that frequently the nerve-trunks become similarly implicated.

PROGNOSIS.

The power of regeneration of nerve-fibres seems almost unlimited, the length of time required for the completion of the regenerative process varying from a few weeks to seven years or more. Poore[25] refers to a patient with traumatic paralysis of the brachial plexus, accompanied by vaso-motor and trophic changes, who had almost completely recovered after four years and a half.

In one of Trepte's[26] cases of neuritis migrans, recovery took place at the end of seven years.

The probable duration and progress of the case can only be determined with any degree of accuracy by careful and repeated electrical examination.

The electrical reactions in the nerves and muscles, as pointed out by Erb,[27] are of the greatest prognostic significance. He says: "Under otherwise similar circumstances, *i. e.*, in one and the same form and cause of disease, the lesion is so much more serious, the duration of the disease the longer, the chance of complete restitution slighter, the more developed and complete the De R. is, and the more advanced the stage in which it is found. Partial De R. is therefore more favorable than the complete, the later stages more unfavorable than the earlier.

In the cases of brachial plexus paralysis reported by Remak,[28] the brachialis anticus and biceps recovered first and the supinator longus last. He is therefore of the opinion that this may perhaps be dependent upon the length of the nerves, which must be regenerated from the point of the lesion.

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[25] Lond. Lancet, 1881, p. 495.

[26] Casuistische Beiträge zur Lehre von der Neuritis, besonders der Neuritis traumatica und migrans. Inaug. Dissert., Halle, 1886.

[27] Handbook of Electrotherapeutics, 1883, p. 86.

[28] Berlin. klin. Woch., No. 9, 1877.

Erlenmeyer[29] takes exception to these views, maintaining that the time of regeneration depends upon the degree of the paralysis and the amount of the structural changes, as deduced from the character of the electrical reaction by which such conditions are determined. In support of this assertion he cites the first case of M. Bernhardt,[30] where recovery occurred first in the biceps and brachialis anticus, next in the supinator, later in the deltoid, and last in the supraspinatus and infraspinatus. The correctness of the views of Erb and Erlenmeyer is clearly demonstrable in our case. The biceps group, presenting *partial De R.*, recovered first, while the supinator longus and the extensors of the forearm, exhibiting *complete De R.*, were the last to show signs of improvement.

The pathology of peripheral neuritis is so well known, that I have purposely refrained from its discussion.

Whether the inflammatory process in this case was of an interstitial or parenchymatous character, or both combined, is of little or no practical importance.

The time required for the regeneration of the nerve-fibres is necessarily influenced by the character and severity of the lesion, by the recuperative powers of the individual, and occasionally by treatment. Recovery sometimes occurs spontaneously, *i. e.*, without the aid of therapeutic measures. In the present case it can be safely assumed that, after the acute symptoms had subsided, the patient neglected all further treatment.

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[29] Corresp.-Bl. d. Schweizer Aerzte, 1882, V., 12, p. 619

[30] Zeitsch. für klin. Med., Bd. IV., 1882, p. 415.